MANAGEMENT OF PORTAL HYPERTENSION*

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I CONSIDER it a privilege to speak to you on the subject of portal hypertension and, more specifically, on the clinical management of cases that exhibit the syndrome. It is a subject of particular interest to me, and I feel most fortunate that my association with Dr. A. H. Blakemore has enabled me to become an observer, a recorder, and in a small way, a contributor to the enormous amount of pioneer work that he has done in this field.

A transient elevated pressure within the portal system exists in many normal physiological states; for example, in direct response to histamine or epinephrine, in response to the Valsalva maneuver or following hepatic plexus stimulation. A reversible elevated pressure also exists in many pathological states, such as congestive heart failure, bronchial asthma and pericardial effusions. However, the irreversible pathological states of constrictive pericarditis, inferior vena cava, hepatic vein, intrahepatic and extra-hepatic venous obstruction are of particular interest to the physician because of the usually striking anatomical deformities which lead to the diagnosis of portal hypertension. The surgeon shares an equal interest because of his increasing capacity to correct some of its causes or results.

This evening I wish to limit my discussion to the most frequently encountered form of portal hypertension, that which is secondary to an intrahepatic venous block caused by portal cirrhosis. It is generally believed that hypertension is due to a deformed intrahepatic vascular anatomy, which is caused by hepatic cell death followed by irregular and eccentric nodular regeneration. The expanding nodules obliterate or compress some of the sinusoids and portal vein radicals, while other sinusoids dilate and bring about ease of communication between hepatic

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TABLE I—PORTACAVAL SHUNT FOR PORTAL HYPERTENSION
1943-1958

Intrahepatic Block	318
Attempted	8
Extrahepatic Block	70
Attempted	6

arterioles and portal vein branches. Through death of the lobule and eccentric regeneration, the central vein comes to be situated near the periphery of the nodule; therefore, portal venous and hepatic arterial blood can pass directly to the central vein and thus by-pass much of the parenchyma. These vascular changes, summarized by Popper & Shaffner[1], are of particular importance because they set the stage for portal hypertension and for diversion of the blood supply from the regenerating hepatic cell. Hemorrhage from varices is likely, and each hepatic cell is on the brink of anoxia.

General as these terms have been, they govern much in our philosophy of management and explain why we are reluctant to subject a patient to the stress of an operation following a shock-producing hemorrhage; why we are anxious to stop hemorrhage and not just keep up with it by transfusion; and why we are not surprised to see evidence of diminished liver function following relatively minor bleeding from varices.

The bulk of experience which I wish to summarize has been derived from a series of 318 cases collected from 1943 to 1958 (Table I). From this point on I should like to take up four major management problems in the same sequential order in which they frequently confront the physician.

Hemorrhage from Esophageal Varices: Bleeding from varices is not always large in amount, but frequently it is, and at such times it requires the facilities of a hospital for control. The diagnosis of bleeding from varices is not always easy to make, since the bleeding must be differentiated from that of peptic ulcer. In our overall series there has been a coincident ulcer or ulcer history in 25 per cent of the cases, and for this reason an esophagram on an emergency basis is undertaken if the

TABLE II—HEMORRHAGE CONTROL—60 CONSECUTIVE CASES
1956-1958

Current Bleeding			25
	No Ascites	Balloon Stopped	6
		Balloon Failed to Stop	3
		Stopped Spontaneously	3
	Ascites	Balloon Stopped	3
		Balloon Failed to Stop	10

patient's condition permits. The esophageal balloon can be used as a differential diagnostic tool to aid in distinguishing esophageal from gastric bleeding. Often this is a clear-cut separation, but it is by no means absolute. General support in the form of oxygen, blood, and bed rest is given and, if there is evidence of continued bleeding, balloon tamponage is favored as a method of control.

Linton[2] passes a large balloon into the stomach, inflates it by a double-lumen tube and applies two pounds of traction for 8 to 12 hours, during which time the blood volume is restored and the patient is prepared for transthoracic esophagotomy and ligature of varices. All patients are committed to operation. Linton reports an 80 per cent success rate, but he does not delineate the severity of the disease in which this result was achieved.

Blakemore advocates passage of a double balloon, triple-lumened tube for compression tamponage. He thereby avoids committing all patients to immediate operation and reserves operative ligation for those who fail to be controlled by compression alone.

The esophageal balloon tube is a useful, life-saving but treacherous device. We are fully aware of its capacity to lead to aspiration, asphyxiation, and esophageal erosions. Most of these tragedies can be averted only through strict attention by the physician and watchfulness on the part of specifically trained personnel. A recent series reported by Conn[3] serves to emphasize that, if inadequately supervised, compression tamponage by the esophageal balloon can lead to serious complications, many resulting in death.

A series of 60 consecutive cases was selected because of uniformity

TABLE III—DEFINITIVE THERAPY—60 CONSECUTIVE CASES
1956-1958

Current Bleeding	25			*Deaths*
No Ascites	12	PC Shunt	12	1
Ascites	13	PC Shunt	3	0
		Varices Suture	2	0
		No Operation	8	8
Previous Bleeding	35			
No Ascites	26	PC Shunt	26	1
Ascites	9	PC Shunt	9	2

of management (Table II). Balloon tamponage failed to stop bleeding in roughly one-half the cases that were actively bleeding on admission to the hospital. Our experience has undoubtedly been shared by other surgeons and has led to other therapeutic attempts, all operative. Specifically, I am referring to segmental gastric and esophageal resections and the emergency portacaval shunt. Although a substantial percentage of patients survive these operations, it is highly unlikely that this percentage will approach that of patients who survive a portacaval shunt electively performed after hemorrhage has been conservatively controlled. If these procedures are reserved for the individuals in whom hemorrhage cannot be controlled, the risks would be prohibitive. For these reasons, we currently advocate early esophageal balloon tamponage without traction; and if this fails to stop bleeding, we recommend simple gastrotomy with direct suture of gastric varices, the usual source of bleeding in instances where esophageal balloon tamponage has failed (Table III).

During episodes of massive bleeding from varices, coma is a common complication. Although we are reluctant to accept the ammonia intoxication syndrome as the sole etiological mechanism, the concept has provided certain valuable therapeutic tools. Mechanically ridding the gastrointestinal tract of substrate for bacterial action and modifying bacterial activity by antibiotics can produce a dramatic awakening in many instances. The use of glutamic acid or arginine has, in our ex-

perience, been of little proven value. Our impressions have been comparable to those recently reported by Reynolds, Redeker, and Davis[4].

Preoperative Management and Case Selection: Assuming that hemorrhage has been brought under control, the next step is to prepare the patient for some therapeutic measure that will prevent repetition of hemorrhage. Although the precise mechanism of initiation of bleeding from varices is not always known, it has been observed that once an individual case has bled, repetition is likely. Preparation of the patient is synonymous with a strict medical program for the treatment of cirrhosis; and to date, that which has been set forth by Patek has been the most acceptable. In 1953, Blakemore and Fitzpatrick[5] set forth what they felt were critical levels of liver function studies dividing the good from the bad surgical risks. The good risk patient had a cephalin flocculation of 2 + or less, a BSP retention of 30 per cent or less in one half hour, a serum albumin of 3.5 gm. per cent or more and a serum bilirubin of 1.5 mgm. per cent or less. During the past three years an addition has been made to these criteria which seems to be of paramount importance. If, while on a well-controlled medical regimen in a hospital, the patient can demonstrate the capacity to improve a depressed level of liver function or to sustain a high level, then the postoperative mortality will be less than 5 per cent.

It is, therefore, our practice to advise definitive surgical therapy for only those cases in which a reserve capacity of the liver can be demonstrated and to discourage surgical therapy for those cases in which liver function continues to deteriorate despite a good medical regimen.

Selection of Operation: At present there are three general classes of operations for the control of bleeding from gastric and esophageal varices:

1. Interruption of the blood supply to the varices.
2. Excision of part or all of the varices-bearing segment of the upper gastrointestinal tract.
3. Decompression of the portal venous bed by a portacaval shunt.

In regard to interruption of the blood supply to the varices, be it accomplished by direct suture as advocated by Crile[6] or blood vessel interruption as advocated by Tanner[7], control of bleeding is at best temporary. Excision of part or all of the varices-bearing segment of the upper gastrointestinal tract can be an effective method of controlling hemorrhage. The main disadvantage is that it is frequently attended by

TABLE IV—EXPECTED SURVIVAL FOLLOWING PORTACAVAL SHUNT
(Operative deaths excluded)

Year	*Approx. % Alive*
1st	90%
2nd	60%
3rd	
4th	
5th	
6th	
7th	30%
8th	20%

nutritional disturbances, undesirable in the long term therapy of cirrhosis. For this reason, we have reserved these procedures for those cases in which a portacaval shunt cannot be performed.

The portacaval shunt, in our opinion, remains the procedure of choice and, of the possible varieties, the end-to-side portal vein-to-vena cava anastomosis is the most uniformly efficient. The instance of subsequent hemorrhage in a series of 318 has been 15 per cent for the portal vein-to-vena cava shunt and 45 per cent for the splenorenal shunt. A significant number of the failures of portal vein-to-vena cava shunt can be attributed to mechanically poor anastomoses resulting from an unusually small or partially thrombosed portal vein. In selected instances, the use of splenoportography, as advocated by Rousselot[8], facilitates the decision as to whether to use the portal or the splenic vein in establishing a shunt.

Follow-Up Care: In any given case where acute massive hemorrhage has been controlled by tamponage and where the patient has been successfully prepared for and carried through a portacaval shunt, we believe that we have extended life expectancy. By the very nature of the disease process, which encompasses an enormous number of variables, direct comparison with a control series of conservatively treated cases can often be challenged. It is a universally accepted clinical impression that hemorrhage from varices can initiate a chain of events that leads to rapid liver failure and death. Philosophically, any therapeutic mea-

TABLE V—EPISODIC STUPOR—60 CONSECUTIVE CASES
1956-1958

Portacaval Shunts	51		
Survived Operation	47		
Episodic Stupor	19		
Temporary	8	Associated Diabetes	0
		Associated Ascites	1
Prolonged	11	Associated Diabetes	3
		Associated Ascites	3
		Associated Diabetes & Ascites	3
No Episodic Stupor	28	Associated Diabetes	1 (mild)
		Associated Ascites	7 (mild)

sure that controls active homorrhage or prevents future hemorrhages should be of value, if, at the same time, the measure does not accelerate the basic disease process in the liver. Without intent to compare, Table IV will give some idea as to expected longevity following successful portacaval shunt; it includes patients who have had recurrence of bleeding, presumably as a result of a late failure of the shunt to reduce portal pressure to safe levels.

Episodic stupor (the so-called ammonia intoxication) has been advanced by the critics of portacaval shunting as an inherent danger of the procedure. This is true, but as our recognition and experience have been extended, the dangers of episodic stupor have diminished.

Uncontrolled, this complication has probably contributed in the past to a diminished life expectancy following portacaval shunt. However, with the advent of Neomycin and Bacitracin, administered orally in combination and in small doses on a long-term basis, patients have remained free of episodic stupor for as long as two years, despite continued high protein diets (Table V).

Summary: In summary, it is our current conviction that hemorrhage resulting from varices associated with portal hypertension arising from cirrhosis of the liver should be controlled immediately by balloon

tamponage of the esophageal varices. If hemorrhage is not controlled, immediate gastrotomy and ligature of the bleeding points should be undertaken. After a period of preparation on a controlled medical regimen, portacaval shunts should be performed on those patients who have the capacity to improve liver function if previously depressed, or sustain a high level of liver function if initially present. In those instances where a shunt cannot be performed because of portal or splenic vein thrombosis, or has failed, obliterative or excisional procedures should be considered.

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